

WHAT IS CLAIMED IS:

1. A method of protecting an individual from a loss of neurogenesis capacity resulting from neuroinflammation, the method comprising:
contacting said individual with a dose of an anti-inflammatory agent effective to reduce neuroinflammatory activity by recruitment or activation of monocyte/microglial cells;
wherein said loss of neurogenesis capacity is reduced.
2. The method according to Claim 1, wherein said inflammation and activation of microglial cells is the result of cranial irradiation.
3. The method according to Claim 2, wherein said radiation is ionizing radiation.
4. The method according to Claim 3, wherein said contacting is performed prior to said irradiation.
5. The method according to Claim 3, wherein said contacting is performed subsequent to said irradiation.
6. The method according to Claim 1, wherein said anti-inflammatory agent is a non-steroidal anti-inflammatory agent.
7. The method according to Claim 1, wherein said anti-inflammatory agent specifically blocks IL-6 activity.
8. The method according to Claim 1, wherein said anti-inflammatory agent blocks MCP-1 activity.
9. The method according to Claim 1, wherein said loss of neurogenesis capacity is associated with Alzheimer's disease, Parkinson's disease, multiple sclerosis, depression, bipolar disorder or Cushing's disease.
10. The method according to Claim 1, wherein said loss of neurogenesis capacity is associated with Lewy Body dementia, Frontotemporal dementia/Pick's disease, AIDS dementia

complex, dementia puligistica and chronic cognitive dysfunction following head trauma, prion-associated dementia such as Creutzfeldt-Jacob disease, cognitive dysfunction following chronic seizure disorders or an episode of status epilepticus, or cognitive dysfunction following encephalitis or meningitis, amyotrophic lateral sclerosis (ALS)/parkinsonian/ dementia complex of Guam.

11. The method according to Claim 1, wherein said loss of neurogenesis capacity is associated with acute injury.

12. The method according to Claim 1, wherein said loss of neurogenesis capacity is associated with pre- or peri-natal ischemia/hemorrhage associated with the developmental dysregulation of stem/progenitor cells in early life.

13. The method according to Claim 1, wherein said loss of neurogenesis capacity results from the attenuation by inflammation of neurogenesis in response to surgical interventions, injury, or disease.

14. The method according to Claim 1, wherein said neurogenesis is central nervous system neurogenesis.

15. The method according to Claim 1, wherein said neurogenesis is peripheral nervous system neurogenesis.

16. The method according to Claim 15, wherein said loss of neurogenesis capacity is associated nerve injury due to trauma, surgery, cancer, multiple sclerosis, ALS, or other motor neuron disease where endogenous or grafted progenitor/stem cells are influenced by immune mechanisms.

17. A method of protecting an individual from a loss of neurogenesis capacity resulting from neuroinflammation as a result of transplantation of neural progenitors to the central nervous system, the method comprising:

contacting said individual with a dose of an anti-inflammatory agent effective to reduce neuroinflammatory activity by recruitment or activation of monocyte/microglial cells;

wherein said loss of neurogenesis capacity is reduced.

18. A method of screening a candidate agent for modification or detection of stem or progenitor cell dysfunction, the method comprising:

determining the effect of said agent on stem or progenitor cell dysfunction in the absence and presence of an anti-inflammatory agent.

19. A method of screening a candidate agent for activity in protecting an individual from a loss of neurogenesis capacity resulting from neuroinflammation, the method comprising:

contacting a model for neuroinflammation with a candidate agent, and determining the effectiveness of said agent on neurogenesis.

20. A method of screening a candidate agent for activity in protecting an individual from a loss of neurogenesis capacity resulting from neuroinflammation, the method comprising:

contacting a model for neuroinflammation with a candidate agent, and determining the effectiveness of said agent on inhibiting expression of MCP-1.